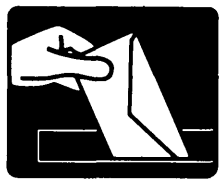
Letters to the Editor and Comments on Practice



Readers will note a change in the title of this section. The intent of this change is to provide a forum for clinical commentary on patient care. As has been the policy with Letters to the Editor, in order to encourage free exchange of ideas, this section will not be peer reviewed. The opinions presented here do not necessarily reflect the opinions of the Editors or the American Diabetes Association.

Teledyne Sleep Sentry: False Security?

I read with interest Hansen and Duck's study entitled "Teledyne Sleep Sentry: Evaluation in Pediatric Patients for Detection of Nocturnal Hypoglycemia" (Diabetes Care 1983; 6:597–600). Although the trend toward tighter diabetes control does necessitate closer observation to avoid the risk of nocturnal hypoglycemia, it appears that those with the highest risk of severe unconscious/convulsive reactions are perhaps being given false security by the instrument described in this article.

The greatest risk associated with nocturnal hypoglycemia is for those who do not awaken before severe neurologic symptoms (e.g., convulsions) occur. According to recent literature,[1,2] this population appears to be characterized by an abnormality in glucose counterregulatory response with decreased epinephrine and glucagon response to decreases in blood sugar. If in fact those at risk do not respond to hypoglycemia with epinephrine response and associated increases in sweating, severe hypoglycemia may occur without warning in those wearing the Teledyne Sleep Sentry. The false security engendered by the device might prevent such patients from taking effective precautions (e.g., extra snacking, testing of blood sugar in early morning, etc.), and thus might in reality even increase the risk of severe hypoglycemic reactions.

Although there is a definite need for an instrument to warn diabetic patients of hypoglycemia during sleep, the present device has a number of limitations including the one outlined above. Patients should be made aware of these drawbacks before use of the Teledyne Sleep Sentry is encouraged.

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REFERENCES

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[2] Boden, G., Reuchard, G. A., Hoeldtke, R. D., et al.: Severe insulin induced hypoglycemia associated with deficiencies in the release of counter-regulatory hormones. N. Engl. J. Med. 1981; 305:1200–1205.

Loading Gauge Safety

"Insulgage," a device manufactured by MEDITEC, INC. (948E E. Orchard Drive, Englewood, Colorado 80110), is a loading gauge that supposedly enables one to draw up a prescribed insulin dosage in a disposable syringe without searching for the correct mark on the measurement scale. The manufacturer claims that by using the "Insulgage" (1) one avoids the possibility of serious human errors, such as setting on incorrect dosage mark and (2) visually impaired diabetic patients gain independence since they now can safely measure their own insulin and self-administer their dosage.

The Maine Center for the Blind has brought to our attention that the measurement of some of the batches of this product is inaccurate, and that an alarmingly high percentage of these devices are incorrectly calibrated. In other words, all the different batches of "Insulgage" do not give the anticipated dosage of insulin.

The Maine Center for the Blind has brought this case to the attention of the Bureau of Medical Services, Food and Drug Administration (FDA), since it seems to represent a deficiency in quality control.

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Hypoglycemia Associated with Trimethoprim/ Sulfamethoxazole Therapy

The ability of various drugs to produce hypoglycemia is well recognized.[1] Sulfa-based antibiotics may potentiate the hypoglycemic effects of sulfonylureas[2] and also may produce hypoglycemia without concomitant sulfonylurea administration.[3] Trimethoprim and sulfamethoxazole are a combination of antimicrobial agents that has been widely used in the treatment of many infections. This drug combination has been cited infrequently as a potential cause of hypoglycemia.[4,5] Two recent cases that have come to our attention suggest that this antibiotic combination may more commonly contribute to the development of severe hypoglycemia in susceptible individuals than has been previously recognized.

Case 1. An 85-yr-old woman with a history of diet-controlled type II diabetes mellitus was admitted to the Beth Israel Hospital because of the sudden onset of confusion followed by unresponsiveness. One week before admission she developed symptoms of a urinary tract infection and was started on trimethoprim (160 mg)/sulfamethoxazole (800 mg) (Bactrim DS, Roche Laboratories, Nutley, New Jersey) 1 tab PO b.i.d. Her other medications included propoxyphene napsylate/acetominophen (1 tab PO b.i.d.) and occasional salicylate for osteoarthritis. She was not taking insulin or oral hypoglycemic agents. Her dietary intake before admission was reported to be poor.

On admission her pulse was 68 and regular, BP 130/50 mm Hg, T = 99°F, and respiratory rate = 14/min. Positive physical findings included evidence of osteoarthritis and Parkinson's disease. Pertinent laboratory data included a blood glucose of 24 mg/dl, prothrombin time of 18.2 s over a control of 12.0 s, and an albumin of 2.8 g/dl (nl 3.4–5.0). BUN was 17 mg/dl (nl 8–20) and the creatinine was 1.3 mg/dl (nl 0–1.3). SGOT, SGPT, alkaline phosphatase, and bilirubin were normal. The serum insulin level was 3 μU/ml and serum cortisol was 50 μg/dl (nl < 25) when the blood glucose was 24 mg/dl. The patient responded promptly to the rapid administration of 25 g of intravenous glucose. Bactrim was discontinued and she was treated with intravenous glucose at the rate of 5 g/h (1.6 mg/kg/min), intramuscular injections of vitamin K, and a 1000-kcal American Diabetes Association (ADA) diet. Eight hours after admission, while receiving intravenous glucose at a rate of 5 g/h, the blood glucose was 56 mg/dl. All subsequent blood glucose measurements were normal. The prothrombin time became normal on the fourth hospital day. On the eighth hospital day the patient underwent a 24-h fast without developing hypoglycemia. She was not rechallenged with trimethoprim/sulfamethoxazole. The patient was discharged in good condition on propoxyphene napsylate/acetominophen 100 mg PO b.i.d.

Case 2. A 74-yr-old woman with a history of diet-controlled type II diabetes mellitus was admitted to the Beth Israel Hospital following loss of consciousness. Her past medical history included thyrotoxicosis treated with RAI 6 yr previously, nephrolithiasis, and chronic renal failure. One week before admission she developed symptoms of a urinary tract infection and was started on trimethoprim (160 mg)/sulfamethoxazole (800 mg) (Bactrim DS) 1 tab PO b.i.d. Other medications included digoxin 0.125 mg PO q.d. and ferrous sulfate 325 mg PO t.i.d. Her dietary intake before hospitalization was reported to be poor. On physical examination her pulse was 80 and regular, BP 144/60 mm Hg, T = 99°F, and respiratory rate = 16. The patient was dehydrated and obtunded. Admission blood glucose was 12 mg/dl, BUN 141 mg/dl, creatinine 8.2 mg/dl, sodium 126 meq/L, and albumin 2.9 g/dl. PT, SGOT, SGPT, alkaline phosphatase, and bilirubin were normal. The patient responded promptly to the rapid infusion of 50 g of intravenous glucose. The trimethoprim/sulfamethoxazole was discontinued and she was treated with D_5W at the rate of 5 g/h (2.1 mg/kg/min) and an ADA diet. Eight hours after admission blood glucose was 38 mg/dl and the D_5W infusion rate was doubled. All subsequent blood glucose concentrations were normal. The serum insulin level was 6 μU/ml, the serum cortisol was 33.5 μg/dl, and the serum alanine level was 226.22 nM/ml (nl > 200 nM/ml) when the blood glucose concentration was 12 mg/dl. The BUN and creatinine stabilized at 58 and 4.1 mg/dl, respectively, and the serum sodium became normal. The patient was discharged in good condition on digoxin 0.125 mg 3 times/wk.

Comments. Hypoglycemia may result from either decreased glucose production or increased glucose utilization. Both subjects in this report had a history of type II diabetes mellitus and poor nutrition before admission. Although hepatic glycogen stores were not assessed in either subject, they can be presumed to be decreased, especially in case 1, who had other evidence of a decrease in hepatic synthetic activity (low PT). On the other hand, neither subject had evidence of adrenal insufficiency that may have contributed to the hypoglycemia. In subject 2 there was no evidence of a decrease in alanine availability as a gluconeogenic precursor as has been described in other subjects with hypoglycemia and renal failure.[6]

In both subjects, an inappropriate increase of glucose utilization appears to have contributed to the hypoglycemia. Subject 2 developed a second episode of hypoglycemia during glucose infusion at a rate greater than her estimated basal glucose utilization; subject 1 required an increase in glucose infusion rate 8 h after admission for a falling blood glucose level. In both subjects, hypoglycemia was temporally related to the institution of therapy with trimethoprim/sulfamethoxazole. The intake of salicylates[7] in the first case and coexisting renal failure[8] in the second case may have made these subjects more susceptible to the hypoglycemic effect of trimethoprim/sulfamethoxazole.